In the claims:

This listing of the claims will replace all prior versions and listings of the claims in the application:

1. **(Currently amended)** A method of reducing fibrosis ~~treating a non-neoplastic hyperproliferative cell or excessive cell accumulation disorder~~ in a patient in need thereof, said method comprising administering to said patient ~~a therapeutically~~ an effective amount of an EphA2 agonistic agent, wherein said EphA2 agonistic agent binds EphA2 and increases EphA2 cytoplasmic tail phosphorylation, increases EphA2 autophosphorylation, increases EphA2 degradation, reduces a pathology-causing cell phenotype, or reduces EphA2 activity wherein said activity is not autophosphorylation.

2. **(Currently amended)** The method of claim 1 wherein said ~~non-neoplastic hyperproliferative cell or excessive cell accumulation disorder is a hyperproliferative epithelial cell~~ fibrosis is a disorder selected from the group consisting of ~~asthma, chronic pulmonary obstructive disease~~, lung fibrosis, asbestosis, IPF, DIP, UIP, kidney fibrosis, liver fibrosis, and other fibroses~~, bronchial hyper responsiveness, psoriasis, and seborrheic dermatitis~~.

3. **(Currently amended)** The method of claim 2, wherein a pathology-causing cell phenotype of said ~~hyperproliferative epithelial cell~~ disorder is secretion of mucin, differentiation of an EphA2-expressing cell into a mucin-secreting cell, secretion of inflammatory factors, or epithelial or endothelial cell hyperproliferation.

4. - 6. **(Canceled)**

7. **(Original)** The method of claim 1 wherein said EphA2 agent is an antibody or antigen binding fragment thereof.

8. **(Canceled)**

9. **(Original)** The method of claim 7 wherein the said antibody is a monoclonal antibody.

10. **(Original)** The method of claim 9 wherein said monoclonal antibody binds EphA2 with a K_{off} of less than $3x10^{-3}s^{-1}$ under conditions appropriate for antibody-EphA2 binding.

11. **(Currently amended)** The method of claim 9 wherein said monoclonal antibody is Eph099B-102.147, Eph099B-208.261, Eph099B-210.248, or B233. ~~or comprises a CDR from Eph099B-102.147, Eph099B-208.261, Eph099B-210.248, or B233.~~

12. **(Original)** The method of any of claims 7, 9, or 10 wherein said monoclonal antibody is a human antibody.

13. **(Original)** The method of any of claims 7, 9, 10, or 11 wherein said monoclonal antibody is humanized.

14. **(Original)** The method of claim 1 wherein said administration increases EphA2 phosphorylation in a treated cell relative to the level of EphA2 phosphorylation in an untreated cell.

15. **(Canceled)**

16. **(Original)** The method of claim 1 further comprising the administration of one or more additional non-neoplastic hyperproliferative cell or excessive cell accumulation disorder therapies.

17. **(Original)** The method of claim 16, wherein said pathology-causing epithelial or endothelial cell phenotype is secretion of mucin, differentiation of an EphA2-expressing cell into a mucin-secreting cell, secretion of fibronectin, secretion of inflammatory factors, or epithelial or endothelial cell hyperproliferation.

18. – 26. **(Canceled)**

27. **(Currently amended)** The method of ~~any of~~ claims 1~~, 15,~~ or 17 further comprising the administration of one or more immunomodulatory agents.

28. **(Original)** The method of claim 27 wherein said immunomodulatory agent is an antibody that immunospecifically binds IL-9.

29. – 32. **(Canceled)**

33. **(New)** A method of reducing fibronectin expression in a cell comprising contacting said cell with an EphA2 agonist, wherein said agonist is an EphA2 antibody or antigen binding fragment thereof.

34. **(New)** A method of reducing secretion of inflammatory factors from a cell comprising contacting said cell with an EphA2 agonist, wherein said agonist is an EphA2 antibody or antigen binding fragment thereof.

35. **(New)** The method of claim 34 wherein said inflammatory factors are IL-8 or IL-6.

36. **(New)** A method of reducing levels of EphA2 in a cell comprising contacting said cell with an EphA2 agonistic agent, wherein said EphA2 agonistic agent binds EphA2 and increases EphA2 cytoplasmic tail phosphorylation, increases EphA2 autophosphorylation, or reduces EphA2 activity wherein said activity is not autophosphorylation.

37. **(New)** The method of claim 36 wherein said agonistic agent is an EphA2 agonistic antibody or antigen binding fragment thereof.

38. **(New)** The method of claim 36 wherein said cell overexpresses EphA2.

39. **(New)** A method of increasing cell-cell adhesion of a cell comprising contacting said cell with an EphA2 agonist, wherein said agonist is an EphA2 antibody or antigen binding fragment thereof.

40. **(New)** The method of claim 39 wherein said cell overexpresses EphA2.

41. **(New)** The method of any of claims 33-40 wherein said cell is a non-neoplastic hyperproliferative cell.

42. **(New)** A method of reducing a pathology-causing cell phenotype of a non-neoplastic hyperproliferative cell, said method comprising administering an effective amount of an EphA2 agonistic agent, wherein said EphA2 agonistic agent binds EphA2 and increases EphA2 cytoplasmic tail phosphorylation, increases EphA2 autophosphorylation, increases EphA2 degradation, or reduces EphA2 activity wherein said activity is not autophosphorylation.